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Inhaled beclomethasone dipropionate (BDP) prevents seasonal changes in atopic asthmatics.

Ponticiello A, Vatrella A, Parrella R, Romano L, Zofra S, Berlingieri GM, Bariffi F.

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University of Medicine Federico II, Institute of Respiratory Diseases, Naples, Italy.

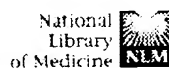
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Inhaled corticosteroids are most effective drugs currently available for the treatment of bronchial asthma. They have been shown to reduce airway inflammation and hyperresponsiveness. The aim of this study was to assess the preventive effect of inhaled steroid therapy in seasonal asthma. In a double-blind study, two groups of 10 allergic asthmatics were randomly assigned to receive inhaled beclomethasone dipropionate (BDP), 500 micrograms b.i.d., or a matched placebo, two puffs b.i.d. The patients used inhaled salbutamol as needed. At the beginning of the study, and every month between February and June, the following parameters were assessed: lung function (forced expiratory volume in one second (FEV1); airway responsiveness (provocative dose of methacholine producing a 20% fall in forced expiratory volume in one second (PD20)), serum eosinophil cationic protein (ECP); and blood eosinophil count. All subjects recorded daily asthma symptoms, beta 2-agonist consumption and peak expiratory flow (PEF) values. In the placebo group, all parameters except FEV1 worsened significantly during the pollen season compared with preseasonal values ($p < 0.001$). BDP produced complete protection, although a slight change from baseline was found for symptom score ($p < 0.01$), beta 2-agonist consumption ($p < 0.01$), and eosinophil number ($p < 0.05$) in May, when the pollen load was highest. These data provide evidence that beclomethasone dipropionate treatment is able to inhibit the seasonal changes occurring during natural exposure in asthmatics. This preventive effect is probably due to the anti-inflammatory action of beclomethasone dipropionate, as documented by its effect on serum markers of airway inflammation.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial

PMID: 9203805 [PubMed - indexed for MEDLINE]



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Bronchial hyperresponsiveness, hypersensitivity to analgesics and urinary leukotriene E4 excretion in patients with aspirin-intolerant asthma.

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Yoshida S, Nakagawa H, Yamawaki Y, Sakamoto H, Akahori K, Nakabayashi M, Sakamoto M, Hasegawa H, Shoji T, Tajimab T, Amayasu H.

Institute for Comprehensive Medical Sciences, Fujita Health University School of Medicine, Toyoake, Japan. syoshida@nisiq.net

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This study was designed to investigate the protective effect of cromolyn sodium on airway sensitivity to sulpyrine, and bronchial responsiveness to methacholine, and to investigate whether this protective activity is associated with reduction in aspirin-induced excretion of urinary leukotriene E4 (u-LTE4), a marker of the cysteinyl LT overproduction that participates in the pathogenesis of aspirin-induced asthma. We assessed the effects of pretreatment with cromolyn sodium on bronchoconstriction precipitated by inhalation of methacholine and sulpyrine in 16 adult patients with mild or moderate aspirin-intolerant asthma; those who were in stable clinical condition and were hypersensitive to a sulpyrine provocation test were included in this study. A double-blind, randomized, crossover design was used. u-LTE4 was measured using combined reverse-phase high-performance liquid chromatography enzyme immunoassay. Cromolyn sodium protected against analgesic-induced bronchoconstriction through mechanisms that are not related to its bronchodilator property, but to the improvement of both bronchial hyperresponsiveness and hypersensitivity to analgesics ($p < 0.01$ and $p < 0.001$). Although excretion of u-LTE4 did not increase after the methacholine provocation test, it significantly increased after sulpyrine provocation ($p < 0.01$). Furthermore, after pretreatment with cromolyn sodium, the maximum level of u-LTE4 after the sulpyrine provocation test was significantly lower than in controls ($p < 0.01$). These results support the hypothesis that cysteinyl LT is one of the most important components in the pathogenesis of aspirin-intolerant asthma. Cromolyn sodium improves both hypersensitivity to analgesics, and bronchial hyperresponsiveness in aspirin-intolerant asthma.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial